SOME COMPLICATIONS OF CHRONIC ENDARTERITIS.

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Fellow of the American Academy of Medicine; Chief of Clinic for Throat and Chest Diseases, University of Maryland, Baltimore.

(Read as an admission paper before the Baltimore Academy of Medicine, April 19, 1887).

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The diagnosis of endarteritis chronica deformans, at its onset, is perhaps made only when a sudden complication makes it too apparent. As to its probable cause—whether gout, syphilis, chronic lead-poisoning, abuse of alcoholic drinks, chronic narcotism, etc.—we are often unable to speak with certainty for a single one. In its varied course and insidious approach, chronic endarteritis makes itself known by the hardness, inelasticity, and tortuousness of the superficial arteries, slowly creeping on from centre to periphery of the vascular system; or, its progress backward to the heart may cause another train of symptoms. In the latter case the sclerosis of the aorta becomes a

Sclerosis of the aortic valves, and the attention is attracted by a systolic or diastolic murmur heard over the aortic area, and by an enlarged left ventricle. In the great majority of cases the important etiological factor in acute endocarditis is acute articular rheumatism, and, indeed, so often is this the case, as we know, that one of the first questions asked of a patient with valvular trouble is, if acute rheumatism has ever preceded this attack of heart disease. But the two important points in the diagnosis between endocarditis from acute rheumatism, and endocarditis from endarteritis is that in the former the mitral valve is by preference affected, and in patients generally under forty years of age; whereas in the latter case chronic endarteritis attacks those over forty, and spreads by contiguity from the aorta to the nearest valve, the aortic, and even if the mitral valve is here affected, it is in company with the aortic valve, whereas after acute rheumatism the mitral is often alone affected.

Another complication is an affection of the aortic valve, due to sudden over-distention of the heart. Peacock[1] made the first important communication on this point. He reported cases of incompetency of the valves from hard work, stretching the valves, as well as cases of direct injury from sudden strain. Albutt[2]

[1] Peacock, Thos. B.: On the Prognosis in Cases of Valvular Diseases of the Heart. London, 1877.

[2] Albutt, Thomas Clifford: Ueber die Folgen der Einwirkung von Gewalt und Ueberanstrengung auf das Herz und die grossen Blutgefässe. London, 1873.

and, later, Myers[1] published papers on the same subject, from cases observed among soldiers. In many treatises, monographs, and works on diseases of the heart, this subject has received scarcely more than a mention. Seitz,[2] Foster,[3] Fraentzel,[4] DaCosta,[5] Flint,[6] Quain,[7] and a few others have given more or less detailed accounts of this affection, but it was not until this past year, when Leyden[8] published his painstaking and interesting article, that the subject has been exhaustively treated. He says that the connection between arterio-sclerosis and bodily overexertion has not been proved, but it is very probably true.

The working classes are particularly prone to this, and the men naturally more than the women. In this case the stiffness and incontractibility of the sclerosed vessel offers too much resistance to the column of blood thrown on it during a violent exertion, and the blood being forced back into the already full left ventricle, causes a sudden distention, with rupture of the aortic valves. In this case we find insufficiency, and occasionally stenosis, of these valves.

A third and frequent complication of arterio-sclerosis is aneurism. This complication may be a sequela of the preceding one. The aorta is atheromatously degenerated, and the left ventricle is hypertrophied. In this condition of things a powerful effort of the hypertrophied left ventricle forces the blood through the aortic valves and upon the aorta, thus causing an aneurism. And even before the sclerosis has extended to the heart, a sudden and violent exertion, as in the majority of cases, causes an aneurism in the atherosed aorta in that part which, by virtue of its position has the greatest strain brought upon it.

The exact definition of the term aneurism is not always clear. Richet[9] says that many aneurisms are due to a dilatation of the vessel without a pathological change in the coats, and that the destruction of the sac does not take place until a long time after the distention.

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[1] Ueber die Häufigkeit und die Ursache von Herzkrankheiten bei Soldaten. London, 1870.

[2] Seitz, J.: Die Ueberanstrengung des Herzens. Berlin, 1871.

[3] Foster, Balthazer: Clinical Lectures on Rupture of the Aortic Valves from Accident, Medical Times and Gazette, December 13, 20, 1873.

[4] See American Journal of the Medical Sciences, January, 1886, p. 97. Quoted by Delafield.

[5] Da Costa: Memoirs of United States Sanitary Commission, p. 372, American Journal of the Medical Sciences, 1871.

[6] Flint: Practice of Medicine.

[7] Quain; Lumleian Lectures, Medical Times and Gazette, 1872, p. 352. Quoted by Delafield.

[8] Leyden, E.: Ueber die Herzkrankheiten in Folge von Ueberanstrengung, Zeitschrift für klinische Medicin. Band XI., S. 105.

[9] Richet, Jaccoud: Nouv. Dictionnaire de Med. et de Chir. prat., t. ii.

Cornil and Ranvier,[1] on the other hand, carefully point out that it is not the result of a single mechanical action upon a healthy vessel which caused the distention, but that these aneurisms are always developed in vessels which have been for a long time the seat of inflammation. Dr. Wilks[2] considers the terms "aneurism" and "dilatation" as expressions of degree, and also seems to be of the opinion that the larger vessels are generally first diseased and then distended, and this without any reference to the number of coats involved.

The microscopical examination of the aneurismal walls of the aorta does not always give satisfactory results. The three coats are supposed to take part in the formation of an aneurism. Ranvier and Cornil,[3] however, have stated that an aneurism of the aorta consisted of the interna and adventitia only, the media having entirely disappeared. I have noticed in specimens that the three coats could be recognized in that part of the artery bordering on the aneurism, but in the aneurism itself the separation between the coats was not clearly marked, notwithstanding the fact that in one specimen the aneurism had just begun to form, the patient having died of an intercurrent disease. Birch-Hirschfeld[4] says that on the border of an aneurism of the aorta the three coats can be distinguished, but toward the middle of the tumor the media disappears first and then the interna and adventitia cannot be distinguished, and, indeed, there is often only a hyaline layer in place of the three coats.

Having once found a pulsating tumor of the chest, the diagnosis of aneurism is not so difficult, as a non-aneurismal tumor of the chest is extremely rare. The difficulty is to find out its exact site. Da Costa[5] says we can do little more than conjecture the position of a thoracic aneurism, because a tumor within the chest leads to such displacements that its relations to the surrounding parts cannot be clearly ascertained during life. The most valuable information we can obtain is from a study of the physiological changes from the symptoms of disturbed function. Indeed, the correctness of our conclusions will depend almost entirely upon that of our interpretation of these symptoms.

The following case, which came under my care, is interesting on account of some of the signs and symptoms noticed. The

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1 Cornil et Ranvier: Manuel d'histologie pathologique, t. i. Paris, 1884.
2 Fagge, Charles Hilton: Principles and Practice of Medicine, vol. ii., p. 840 ff. Philadelphia, 1886.
3 Ranvier et Cornil: Contributions a l'hist. norm. et path. de la tunique interne des artères et de l'endocarde, Archiv de Physiologie, t. i., p. 551.
4 Birch-Hirschfeld: Lehrbuch der pathologischen Anatomie, Band II., S. 98.
5 Da Costa: Medical Diagnosis, fourth edition, p. 388.

meagreness and incompleteness of the history, however, is due to the fact that the patient failed to reappear, and thus escaped an inevitable post-mortem, which would have confirmed or corrected the diagnosis :

Mrs. B——, aged forty-seven, a pale, spare woman, rather under-nourished, came to me complaining of inability to use her voice, and wished to have a supposed laryngitis and bronchitis treated, which she said she had had for some length of time. She complained of pain in the left side of the chest and left arm, and said that her heart "fluttered" very much and seemed to rise up in her throat when she was excited. She was decidedly short of breath, and spoke scarcely above a whisper. On examination a pulsating tumor was observed at the left sterno-clavicular articulation, and was very perceptible to the eye and hand. The tumor, she said, came suddenly after exerting herself during a convalescence from typhoid fever. The following points were obtained: Pain over the cardiac region, especially on pressure, dyspnœa, dysphagia, occasionally œdema and want of sensibility on the left side, displacement of the trachea to the right side. There was also dilatation of the left pupil, almost complete absence of the right radial pulse, and absence of aneurismal bruit in the right carotid and subclavian arteries, which bruit, however, was audible on the left side. The diagnosis of aneurism was at once made, but the exact site could not with certainty be determined. On palpation and percussion the heart was found to be higher than normal, the apex being in the fourth intercostal space and to the right of the left nipple near the sternum, that is, directly above its normal position. There was apparent to the eye and hand much palpitation. Percussion was extremely painful. On auscultation the heart-sounds seemed to be much quickened, but otherwise normal in character. She had complained for some time of a dry cough and of hurried respiration after the slightest exertion. On questioning her, she said that by her first husband she had had two miscarriages and one child, since dead. She had had sore throat (a not unusual occurrence), and her hair had been coming out. She did not remember having had rheumatism. Although there may not be sufficient proof that her trouble was of syphilitic origin, still, without making a diagnosis as to the cause, iodide of potassium with syrup of the iodide of iron were exhibited in gradually increasing doses. After several weeks under this treatment the mydriasis disappeared, the swelling was apparently reduced in size, and her voice returned for a short time.

The history of the case, unfortunately, stops here, as she passed out of my care while I was absent from the city, and has probably succumbed long since to the aneurism, the exact position of which can be conjectured only. Before reviewing the symptoms and signs present in this case, it might be well to glance at some of the statistics on aneurism.

The following figures from Lisfranc[1] give the number of cases collected by him for the different ages :

Age, Years.	Number of Aneurisms.
13	1
15 to 20	3
20 to 30	17
30 to 40	29
40 to 50	37
50 to 60	17
60 to 70	3
70 to 80	3

Much more numerous are the statistics for the different vessels. Thus, Hodgson[2] reports the following cases :

Ascending aorta, arch, and innominate artery	21
Descending aorta	8
Carotid	2
Subclavian and axillary	5

Of the 551 cases of Crisp[3] there were of the

Thoracic aorta	175
Abdominal aorta	59
Subclavian artery	23
Carotid artery	25
Popliteal artery	137

These were in part distributed as follows :

Age. Years.	
20 to 30	71
30 to 40	198
40 to 50	129

In the London Museum[4] the collection of aneurisms is as follows:

Thoracic artery	207
Abdominal artery	42
Popliteal artery	50
Subclavian artery	12
Carotid artery	9

[1] Lisfranc: Article Aneurysme, du Nouveau Dict. de Med. et de Chir. prat., t.
[2] Hodgson: Treatise on the Diseases of Arteries and Veins. London, 1815.
[3] Crisp: Diseases of the Blood-vessels. London, 1847
[4] London Museum Reports.

In Sibson's[1] collection there were 880 aneurisms of the aorta, 703 of which were of the thoracic aorta and distributed as follows :

	Number of Aneurisms.
Ascending aorta (including 87 at the valves of Valsalva)	193
Arch	120
Descending aorta	72

Lancereaux,[2] Baillu,[3] Hayem,[4] and others all agree that the thoracic aorta is most frequently the seat of aneurism. Crisp[5] also says that the anterior and right side of the aorta is most frequently affected, and that the cause is a sudden and violent contraction of the heart. All authors agree that men are more liable than women to this form of aneurism, and naturally, on account of their occupation. The proportion may be stated as about one woman to eight men.

In reviewing the different signs and symptoms in the endeavor to interpret them, some light may be thrown on the situation of the aneurism.

Pain.—The pain was felt on the left side and down the left arm. It was probably due to the pressure of the aneurism on branches of the brachial plexus, and also to pressure caused by general œdema.

Dyspnœa.—This might be due to several causes : (1) simply to a diminution in the size of the thoracic cavity ; (2) to pressure on the left bronchus or trachea ; (3) to pressure on the recurrent laryngeal nerve of the left side in which case we should have a neurotic dyspnœa. As no laryngoscopic examination was made, it might be supposed that the three causes were present. From her manner of breathing and the size of the aneurism, I am inclined to think that the last two causes were present.

Œdema.—This was undoubtedly caused by pressure on some venous trunk, most probably the innominate vein.

Mydriasis.—The mydriasis was caused by pressure upon a branch of the cervical sympathetic (cilio-spinal). This agrees with several other points in the history which go to show that the aneurism had not grown to a very large size. The fact that the pupil on the left side was dilated would tend to show that

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1 Sibson Medical Anatomy. London, 1869.
2 Lancereaux : Traité historique et pratique de la Syphilis. Paris, 1865
3 Baillu : Morbid Anatomy.
4 Hayem Medical Dictionary, 1839.
5 Crisp : Loc. cit.

the pupil on the left side was dilated would tend to show that the fibres of the cervical sympathetic going to the musculus dilatator pupillæ were simply irritated and not destroyed, in which latter case the pupil would be contracted, since the oculo-motorius and sphincter pupillæ would have no opponent. The cases reported recording an affection of the pupil generally refer to a contraction, and not to a dilatation, of the pupil. Gairdner[1] reported a case of aneurism of the aorta which projected up into the neck, causing a contraction of the pupil on the affected side. But on further noticing this case it seems that the patient was taking large doses of opium, which certainly could not fail to have an effect on the pupil. Robertson[2] reports a case of division of the cervical sympathetic, with contraction of the pupil on the affected side. Dr. John Reid[3] was the first to point out this inequality of the pupils in case of tumors of the neck interfering with the cervical sympathetic.

Hurried respiration.—This was caused by pressure upon the left pneumogastric nerve, which pressure also caused the increased rapidity of the heart-beat.

Dysphagia.—This may be explained on the same ground as the pressure on the trachea.

Want of sensibility on the left side.—This was probably due to the œdema, as well as to the pressure on the sensory nerves.

Almost complete absence of right radial pulse.—This is the most difficult sign to understand. Generally the radial pulse on the affected side is indistinct or absent, and in this case we must either record this sign as an exception which may possibly occur, or try to show that the aneurism extended in some way over to the right, or was actually situated on the right side of the thorax, in spite of all the apparent proofs to the contrary. Crisp[4] says, if there is an obstruction to the circulation in the subclavian or innominate artery, there is usually a difference in the pulse of the two sides of the body, the radial pulse of the affected side being less distinct. If, however, the arteries contain no fibrinous deposit, and their calibre be not diminished, the pulsation on the affected side may be stronger.

The condition of the heart.—The valves were found to be normal, and the ventricles apparently not hypertrophied. This is a rare condition in case of an aneurism of the aorta, especially of the ascending and arched portions. In many of such cases reported, the aortic valve was affected.

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1 Gairdner, W. T.: Edinburgh Medical Journal, July, 1855, vol. i.
2 Robertson, Argyll: Edinburgh Medical Journal, February, 1869.
3 Reid, John: Edinburgh Medical and Surgical Journal, 1841.
4 Crisp: loc. cit., p. 129.

Key[1] in speaking of the reaction of aortic aneurisms on the heart, says he has not yet met with a case in which an aneurism of the aorta produced a hypertrophy of the muscular substance of the left ventricle, and in an another report[2] he found an aortic insufficiency in four cases out of seventeen, and no case of stenosis; and his deduction was that if the valves were healthy the aneurism exerts no influence on the heart.

This review of the signs and symptoms, together with the age of the patient, her history, and the above statistics, would point to an aneurism whose probable seat was in the aorta. As an aneurism at the valves of Valsalva produces so few symptoms, and so easily escapes detection, this part may be eliminated. The ascending aorta may be excluded, because an aneurism here would in all probability be on the right side; and even if an aneurism of the ascending aorta were present, and the aneurismal sac pressed over upon the left side of the heart, causing the symptoms related, there would be a bruit or throb in the right subclavian or carotid, which the history shows was absent. The descending aorta may also be excluded; and as the left side was principally affected, it was most probably (unfortunately it is not certain) an aneurism occurring at the anterior and inferior portion of the left part of the arch, opposite to where the left carotid and subclavian are given off. Of course such a diagnosis can be based upon hypothesis alone.

In looking at the etiology of this case, it would not be right to deduce that, because the iodides caused a general improvement, therefore the cause was specific. That the iodides do cause an improvement in many such cases has been witnessed lately by Huchard,[3] of Paris. He records a number of patients suffering from atheroma of the arteries and chronic arteritis, as well as valvular trouble from atheroma, in all of which amelioration and recovery were brought about by the use of the iodides, and particularly of the iodide of sodium. He says these were true cases of organic heart trouble in patients not anæmic. He also particularly affirms that the iodides, in his experience, are not efficacious in every form of valvular disease, but only in chronic so-called sclerotic inflammation of the lining membrane of the heart and the arteries.

NOTE.—Since writing the above, an article by Beverley Robinson, M.D., on this subject, has appeared in THE MEDICAL RECORD, February 26, 1887.

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1 Key, Axel: Medical Times and Gazette, June 4, 1879, vol. i.
2 Ibid.: Hygeira—Proceedings of the Swedish Society of Physicians, p. 285.
3 Huchard, Henri: Bulletin Général de Thérapeutique, t. cxi., p. 302.